With kind regards

CONCERNING THE ACTION

OF

SALTS OF POTASH, SODA, AND AMMONIA

ON THE

FROG'S HEART.

BY

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PROFESSOR OF MEDICINE AT UNIVERSITY COLLEGE, LONDON,

AND

HARRINGTON SAINSBURY, M.D., M.R.C.P.

Read June 13th, 1882.

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Caustic potash, soda, and ammonia, also certain of the salts of potassium, sodium, and ammonium, were examined as to their action on the frog's heart, and found to affect this differently. A word of explanation as to the plan of the paper will be of service.

The mode of experimentation, and the results obtained with the hydrates and with the iodides, bromides, and chlorides of the above bases are first given. These results are then discussed, and the conclusions drawn. Finally, experiments with the citrates of potassium, sodium, and ammonium are given, and the results contrasted with those obtained with the previous salts.

The object in thus separating the salts was that the chlorides, bromides, and iodides might the better be considered apart from the rest as forming a group by themselves. In the table of quantities this plan has been departed from, the citrates being classed with the above salts; this is for the convenience of comparison and to avoid repetition.

The mode of experimentation was the following:—A Roy's tonometer was employed, the heart was tied on to the cannula as near as possible to, if not in, the auriculo-ventricular groove. (It is not always possible to avoid having a portion of the auricle below the ligature; this will be referred to later on when the experiments are discussed.)

As in previous experiments dried bullock's blood dissolved in water, so as to represent normal blood, was used; this was diluted in the earlier experiments with 2 parts of saline solution, $\frac{3}{4}$ per cent., in the later experiments with $2\frac{1}{2}$ parts. In the former 3 oz., in the latter about $3\frac{1}{2}$ oz. (more exactly 100 cubic centimètres), of this mixture were taken.

Where the action of the drugs was estimated quantitatively the dose was as far as possible kept uniform, and the times of addition also, the dose in question being added every quarter revolution of the cylinder (about 2—$2\frac{1}{2}$ minutes) to the whole mass of circulating fluid. The duration of each experiment was made as near an hour as possible, in order to reduce the error due to natural exhaustion which over lengthened periods would become considerable. The dose in consequence had to be adapted to this period. The tracings read from left to right.

The experiments were made in November, December, 1881, January and the early part of February, 1882.

The drugs were tested in two directions:

1. As to their action on the spontaneous working of the heart.

2. As to their action in modifying the effect of continuous faradisation applied to the heart.

The latter action will be first described, and this requires that the effect of continuous faradisation should be described.

Continued faradisation, provided the strength of the current be sufficient, and the interruptions frequent enough, causes in the normal heart a condition of continuous contraction, *i.e.* spasm, of greater or less extent.

To this persistent spasm—in which *fusion* of rhythmic contractions is the most obvious factor—the term "Tetanus" is here applied. As such it resembles closely the tetanus of a skeletal muscle, though differing in certain points.

Marey ('Physiologie experimentale,' vol. ii, p. 81, 1876[1]) we believe was one of the first to describe tetanus of the heart. He shows that the excitability of the heart is not the same at different moments of a cardiac cycle; thus a minimal stimulation administered during systole will not cause a contraction; the period during which this obtains is called the "refractory phase." The length of this period varies with the strength of the stimulus; the stronger the stimulus the less the duration, till the refractory phase quite disappears. Hence, when a series of *weak* electrical stimuli act on the heart, the greater number occurring during the refractory periods are ineffectual, and the number of systoles is much less than the number of excitations. But if the intensity of the current be increased (the frequency of interruption being unchanged), the refractory period lessening, the number of systoles approaches that of the excitations, and may equal them, a fusion results, *i.e.* a condition of tetanus.

Besides the influence of strength of the stimulus, Marey further shows that whilst cold increases heat diminishes the refractory period.

In our experiments, where continuous faradisation was

[1] Luciani ('Ludwig's Arbeiten,' 1872) had previously drawn attention to persistent spasm—the result of fusion of neighbouring beats—and had employed the word "*tetanus*." He shows that it may result as a first effect of the ligature, and also as a first effect of the circulation of serum.

employed, the cannula on which the heart was tied served as the one electrode, the other electrode pressed against the outer surface of the ventricle, care being taken that contact persisted throughout faradisation. The excitations were very frequent, the hammer of the induction apparatus vibrating rapidly.

The results obtained were in accordance with the previous statements from Marey, and it became clear that *fusion*, complete or incomplete, *i.e.* tetanus, complete or incomplete, could be produced in the following ways, viz.:

1. By lessening the refractory period. Increase of the strength of the current will effect this. Raising the temperature will do the same. Further, certain drugs will do the same.

Even in the cool months of October and November we met with hearts whose irritability was such that they exhibited this fusion from the onset. It was especially to this form of fusion that Luciani drew attention.

2. By prolonging the duration of each contraction. It is obvious that, other things remaining the same, increase in the duration of the beat must favour fusion. Some drugs, *e.g.* soda, ammonia, and its salts, probably act in part in this way.

3. Yet another way is to be noted. Certain drugs, *e.g.* soda, ammonia, potash, antiarin, digitalin, strophanthus, induce a persistent spasm of the ventricle, not, however, by means of a fusion of beats, for these may occur so infrequently as to exclude this mode; in such cases, even when the intervals between the beats are a minute or more, the persistent spasm may last during the whole period of the intervals. This persistent spasm will, *cæt. par.*, tend to fuse together neighbouring beats.

It has been suggested ('Journal of Physiology,' December,[1] 1881) that this spasm may be an increase of the tonus of the heart and allied to contracture.

[1] "Regarding the action of Hydrate of soda, ammonia and potash on the Frog's Ventricle."—Sydney Ringer, M.D.

The effect of temperature was tested, and it was found that whilst heat facilitated the production of tetanus, cold hindered such; these results are confirmatory of Marey's statements as to the refractory period. A difference of temperature amounting to 12° C. demonstrated the above very distinctly.

As to the effect of exhaustion, this does not appear to affect the facility with which the heart may be tetanised.

It is worth while contrasting cardiac tetanus with that of a skeletal muscle. In the latter case a stimulus sufficient to excite a good contraction will, if sufficiently frequently repeated, yield tetanus. In the former, a stimulus capable of causing full contraction of the heart, but not in excess of such strength, will not yield tetanus by repetition, however great the frequency attained. The explanation is to be found in the refractory phase attending each contraction. To get tetanus the strength of the current must be increased, whereby the refractory period is diminished up to extinction.

Diagram 1, fig. I, represents the development of tetanus. The figures above the line indicate the positions of the secondary coil of the Du Bois Reymond.

In A we have incomplete fusion; the frequency, though increased, is insufficient to effect complete fusion.

In B the effect of increased strength of current is seen to have caused greater frequency and completer fusion.

In C a further stage is shown, but here, with the secondary coil at 3, also with the coil at 5 in D, the individual beats can no longer be counted, the line being in parts practically straight.

In addition to increased frequency, both B and C show that the element, persistent spasm, is also present as a factor. The length of line above the trace represents the time of faradic excitation.

Having described the effect of continuous faradisation on the heart, we proceed to describe how this effect is modified under the influence of certain drugs.

The mode of experimentation was the following :—The

effect of faradisation was tried before the addition of the drug, and a certain degree of tetanus being selected to serve as standard, the addition of the drug was commenced; the secondary coil was maintained, of course, stationary, and at definite, equal intervals of time the heart was faradised. The intervals were eight or ten minutes in length, sufficient to allow the residual effects from the preceding tetanus to have passed off.

Hydrate of soda, hydrate of ammonia, hydrate of potash.

Hydrate of soda increases the readiness with which the heart answers to continuous faradisation. Thus, a strength of current capable only of slightly increasing the frequency of the normal beats, therefore short of producing tetanus, caused well-marked tetanus after the addition of soda. In this tetanus all three factors are probably at work, viz. diminished refractory period, persistent spasm (of the nature of tonus), increased duration of each beat. This increased readiness of response occurred without any diminution of the height of the trace. In the doses here used, then, soda increases what may be termed excitability, without affecting contractility (Diagram 1, fig. II, A, B, C).

From these observations, made in November, season (temperature ?) would seem to have a modifying influence. The dose of soda required to produce a given effect here was much larger than in summer, and the diastolic contraction, so well marked in summer, was much less developed, and then only after a relatively large dose.

Hydrate of ammonia.—The excitability to the continuous faradic is considerably increased, whilst the height of the beat is still undiminished. This increased excitability is maintained during the early part of the decline in height, then itself begins to decline, but even to the last the excitability remains, at least, as good as before the addition of ammonia (Fig. III, A, B, C, D).

The modifying action of season was marked here also. In summer a much smaller dose is requisite to develop

well-marked diastolic contraction and the persistent spasm indicated by departure from the base line, which spasm increasing, the ventricle finally stops in strong systole, in which condition it remains a considerable time. In the present series, made during November, the above effects were not strongly marked except a very large dose was administered.

Hydrate of potash.—The readiness to become tetanised is markedly decreased by potash, and a strength of current sufficient to give well-marked tetanus before addition subsequently soon loses its effect. A rather curious effect is further to be noted: not only may such current fail to show any tendency towards producing tetanus, it may actually suppress the spontaneous beats, these beginning again a little time after discontinuance of the faradisation (Diagram 2, fig. IV, A, B, C, D, E, illustrate potash). This suppression of the spontaneous beats is not shown in diagram.

Contrasting the effects of soda, ammonia, and potash, we have, on the one hand, soda and ammonia, which early and for a considerable period increase the excitability of the cardiac tissue, as tested by continuous faradisation; on the other hand, potash, the striking effect of which is a diminution of this excitability from the very commencement. This diminished excitability occurs, even though at the time there exist persistent spasm due to the drug (Fig. IV, C, shows this well).

It may be here stated in general terms that, so far as have been examined, the salts of potash act like the hydrate in the way in which they modify the effect of continuous faradisation, *i. e.* they lessen this from the commencement, and may further cause the suppression of spontaneous beats during faradisation above noted.

In the present series the iodide and citrate were tested on this point; the chloride had been previously examined (see the 'Practitioner' for January, 1882).[1]

[1] "Concerning the action of the Chlorides and Bromides of sodium, ammonium, and potassium on the Frog's Ventricle," by Sydney Ringer, M.D.

The fact of the citrate—an organic salt—conforming with the inorganic salts, the iodide and chloride, renders the presumption that salts of potash generally may behave alike in the above respect not improbable.

The other mode of testing the influence of drugs has now to be considered, viz. "*the action on the spontaneous working of the heart.*"

The drugs tested in this direction were of the bases—hydrate of potash alone—of the salts, the iodides, bromides, and chlorides of sodium, ammonium, and potassium, also the citrates of these three bases. Incidentally the modification of the effect of continuous faradisation was examined in the case of the iodides. The results of the experiments will be first detailed, and then the conclusions given to which these led.

The mode of experimentation has already been stated. The strength of the solutions employed was in each case 10 per cent. The quantities of this used are given in cubic centimètres, and the actual quantity of salt in grammes and grains. The quantity of blood mixture used was throughout 100 cubic centimètres.

The reason why of the bases, potassium hydrate alone was taken was that the tendency which the other bases—soda and ammonia—show to produce arrest of the heart in full systole, does not allow of a quantitative comparison between the drugs, since potash arrests in diastole.

Caustic potash.—The salient feature here noted was the persistent spasm excited, but, disregarding this, the effect on frequency, so far as the primary effect went, was inconstant, sometimes slowing, sometimes quickening, but in all cases marked slowing preceded complete inhibition, which occurred in all six cases, and on an average after 0·6 c.c. (= 0·9 grains) had been added and whilst the ordinate was of good value.

When in the final stage single stimuli caused no response, continuous faradisation was without effect.

With regard to the height of the beat, this was not much affected in the early stage, but chiefly towards the

end, and in particular after complete inhibition. No primary increase in height was noted such as ammonia shows. The persistent spasm or diastolic contraction, which has been previously described ('Journal of Physiology,' Dec., 1881), was here particularly well marked.

The quantities required to arrest the heart were:

Exp. I.—Feb. 7.	Temp. of room 15° C.	Quantity 0·75 c.c.	
„ II.— „ 7.	„ 15·5°.	„	0·85
„ III.— „ 8.	„ 15°.	„	1·05
„ IV.— „ 9.	„ 14·5°.	„	1·05
„ V.— „ 9.	„ 15°.	„	0·95
„ VI.— „ 10.	„ 15°.	„	0·65
			5·3

∴ Average 0·88 c.c. = 0·088 grms. = 1·35 grains.

Iodide of sodium.—The effect on the rhythm observed here was a slight lessening of the frequency of the beats, but in no case were these arrested, so that the beats remained spontaneous to the very end, *i.e.* as long as contractility persisted. The heart is arrested in diastole.

In the final stage, when the beats had quite disappeared or showed only as a faint waviness of the trace, and single induction shocks would either give no response or one only just visible, continued faradisation of the same strength or weaker would cause persistent spasm, reaching a maximum by successive steps, each contraction being piled up, as it were, on the top of the preceding one. (See Diagram 3, fig. VII A.

In connection with this it must be mentioned that occasionally, whilst the heart was beating spontaneously but under the influence of the sodium salt, a somewhat similar piling up was observed, occurring without obvious cause.

This phenomenon has been referred to throughout this paper, and the expression "*piling up*" retained, but it must be stated that this mounting up by a succession of steps is not always seen; the term has, however, been kept, and for this reason it must be remembered that the essential part of the phenomenon is the height attained,

which is far in excess of any individual contraction spontaneous or excited. (See fig. VII, A and B.)

As to the effect on contractility the amplitude of the beat gradually lessens, but in the end stages, when the height has been greatly reduced, the breadth of the trace, *i.e.* the duration of the beat, becomes increased, in some cases very considerably.

Iodide of sodium is very little poisonous, as will be seen from the quantities appended, which were those requisite to stop the heart:

Exp. I.—42 c.c.	Temp. at time of experiment		12·5° C.
„ II.—72	„	„	10·5°
„ III.—48	„	„	10°
„ IV.—54	„	„	9·5°
„ V.—52	„	„	9°
„ VI.—60	„	„	8·5°
328			

Average 54·6 c.c. = 5·46 grms. = 84·24 grains.

Iodide of sodium lessens in a marked degree the excitability to continuous faradisation.

Iodide of ammonia.—This is an unstable salt, readily yielding free ammonia and iodine. A good commercial specimen was used, containing, however, traces of the above.

The effect on rhythm is not marked, spontaneous beats continuing till contractility is nearly, if not quite, destroyed; in this respect ammonium iodide agrees with the corresponding sodium salt. The heart is arrested in diastole.

After the heart has been arrested, neither single induction shocks, nor continuous faradisation, has any effect. In this respect the salt contrasts with sodium iodide.

The heart is arrested by the destruction of contractility, the beats growing weaker and weaker till they finally disappear. There is, however, probably in all cases an early stage during which the ventricle empties itself more completely, *i. e.* a stage of stimulation; this is scarcely appreciable when the ventricle is contracting vigorously before

addition of the drug, but when failing to empty itself quite, the above stage is indicated by increased amplitude of the trace, and with this increased height rounding of the top of the trace is noted. If a large dose be suddenly added, both diastolic contraction and persistent spasm appear. In one case, as the effect of a large dose, spontaneous beats were arrested and replaced by a slowly remitting spasm.

This salt is a far more powerful paralyser of the ventricle than iodide of sodium. Six experiments gave the following numbers :

Exp. I.—4·6 c.c.	Temp. at time of experiment		12° C.
„ II.—4·9	„	„	12°
„ III.—4·2	„	„	13°
„ IV.—3·9	„	„	13·5°
„ V.—4·9	„	„	13·5°
„ VI.—2·1	„	„	14°
24·6			

Average 4·1 c.c. = 0·41 grms. = 6·3 grains.

Iodide of ammonium, like iodide of sodium, lessens in a marked degree the effect of the continuous faradic current. Suppression of the spontaneous beats during faradisation may even be witnessed (see Potash).

Iodide of potassium.—The striking feature in the action of this salt is that suddenly, whilst the height of the trace is still considerable, the spontaneous beats are arrested permanently, though there is still response to electric stimulation. If the addition of the drug be still continued the response, originally having the value of the last spontaneous beats, grows less and less till finally contractility disappears. Preceding the arrest of spontaneous beats these latter grow more infrequent. During lengthened intervals between spontaneous beats, or subsequently to permanent arrest, the less the interval since the last spontaneous or excited beat the stronger must be the excitation to be effectual. This of course between limits, for below a certain strength all excitations would be ineffectual, above a certain strength all would be effectual. The

same applies to a heart temporarily inhibited by the ligature. In fact, the ventricle arrested by iodide of potassium behaves in all respects like such heart.

When the contractility has disappeared to single induction shocks, continuous faradisation is without effect; in this respect it resembles iodide of ammonium, but contrasts with iodide of sodium.

Iodide of potassium further acts powerfully on contractility; this is seen, subsequent to arrest of the spontaneous beats, in the decreasing height of the excited contractions.

The following numbers were obtained:

Exp. I.—2 c.c.	Temp. 13° C.
„ II.—2	„ 15°
„ III.—2·2	„ 13°
„ IV.—2·2	„ 14·5°
„ V.—2	„ 15°
„ VI.—1·9	„ 9·5
12·3	

Average 2·05 c.c. = 0·205 grms. = 3·16 grains.

In its lessening the effect of continuous faradisation of the ventricle, iodide of potassium is similar to iodide of ammonium, but more powerful; as with this latter, in the final stages before complete arrest, the spontaneous beats become arrested during the faradisation, and a considerable interval may follow before the spontaneous beats recommence. In this latter respect it, with the ammonium salt, contrasts with sodium iodide.

A character shown in common by the iodides of all three bases was that, subsequently to the arrest of the ventricle by the drug, and when single electric shocks were without effect (also continuous faradisation in the case of ammonium and potassium iodides), the dilution of the circulating fluid with water (100 c.c. were in each case added) brought back spontaneous beats of good value; the recovery was but temporary, but in some cases a second and a third dilution (100 c.c.) were attended with

recovery, each time feebler and of shorter duration. With each recovery a certain amount of persistent spasm occurred, indicated by the trace retreating from the base line.

Bromide of sodium.—Six experiments were made. The effects were somewhat inconstant and anomalous; thus in two cases there was no inhibition whatever, the beats remaining spontaneous till contractility disappeared. These, then, were in accordance with the iodide and citrate of soda series. In one there was distinct inhibition, as marked as in the case of potassium salts. In the remaining three the rhythm was curiously affected, *e.g.* after the quantities had reached 15 c.c., 15 c.c., and 18 c.c. respectively, patches of beats separated by long intervals occurred, the end patch was of long duration, the beats frequent, and the rhythm persisting till contractility had disappeared. Though this phasic condition represents a form of inhibition, still it was not complete inhibition, and in five out of the six the final stages contrasted as markedly with the effects of potassium salts as did those of the other sodium salts.

In the final stage, when the strongest current excited either no response or only the faintest, the characteristic piling up was obtained with continuous faradisation. There was nothing special as to contractility. In the one instance of marked inhibition there was scarcely any reduction in height when this occurred.

The quantities were:

Jan. 19	Temp. 14·5°	...	42 c.c.
„ 24	„ 18°	...	32
„ 24	„ 17·5°	...	41
„ 26	„ 17°	...	34
„ 27	„ 17°	...	28
„ 27	„ 17°	...	24
			201

Average 33·5 = 3·35 grms. = 51·7 grains.

Bromide of ammonium.—The results obtained were very

uniform. In all, frequency was increased; in five, this occurred from the very beginning of addition of the drug, in one, rather marked slowing followed the first dose, but soon gave way to increased frequency. The increased frequency was maintained up to the end, disregarding some slight irregularity which in three cases appeared in the final stage.

Subsequent to the disappearance of spontaneous beats, there was no response to single shocks, and none to continuous faradisation.

On contractility, a primary effect in the way of increase of the height of the beat with broadening of the apex was distinct in some, very slight in others, but discoverable in all. Slight departure from the base line was also noticed, but not in all. The frequency of beats was too great to admit of diastolic contraction appearing. These last-named effects were early, viz. after the first or second dose. Fig. V A (Diagram 2) illustrates these effects.

The decline in height was rather rapid towards the end.

Quantities:

Jan. 28	. .	Temp. 16·5°	...	Quantity 2·6 c.c.	
„ 28	. .	„ 16°	...	„	2·4
„ 30	. .	„ 16°	...	„	2·2
„ 30	. .	„ 16·5°	...	„	2·4
„ 31	. .	„ 16°	...	„	2·6
„ 31	. .	„ 17°	...	„	2·2
					14·4

Average 2·4 c.c = 0·24 grms. = 3·70 grains.

Bromide of potassium.—In the six experiments made with this drug the effect in the way of inhibition was less marked than was the case with the other salts of potassium examined, viz. iodide and chloride. In general, moreover, there was less uniformity of action in the series.

In three out of the six complete inhibition obtained; in the remaining three, though in the final stages, the inhibition was almost complete, still, an occasional spontaneous beat occurred till the amplitude was practically abolished.

The effect, then, though somewhat less marked here, is still very decided.

In five out of the six cases slowing occurred as a primary effect, contractility suffering but slightly; the subsequent effects on rhythm were very irregular.

As to the effect of continuous faradisation, no piling up of the beats obtained as a final effect.

There is nothing special as to the effect on contractility excepting that the action hereon was not marked early. At the time of complete inhibition the beats were severally reduced to $\frac{3}{5}$, $\frac{1}{2}$, and rather less than $\frac{1}{2}$ their original height.

Quantities employed :

Jan. 19	. .	1·7 ...	Temp. 15°
„ 20	. .	2·1 ...	„ 17°
„ 20	. .	2·4 ...	„ 18°
„ 21	. .	2·8 ...	„ 14·5°
„ 21	. .	2·2 ...	„ 15°
„ 23	. .	1·6 ...	„ 13·5°
		12·8	

Average 2·13 c.c. = 0·213 grms. = 3·28 grains.

Of the bromide series the effect of dilution was tested with the ammonium and potassium salts. Dilution with blood mixture was substituted for dilution with water, 100 c.c. being added. Dilution was tried twice with ammonium bromide with negative result; no recovery occurred. With potassium bromide dilution in three cases brought back spontaneous beats of good height. The blood mixture was added when the ventricle had ceased to respond to the strongest stimulation.

Chlorides of sodium, ammonium, potassium.

Some experiments on these salts made in the summer months have already been recorded.[1] But as season

[1] 'Practitioner,' January, 1882.

(temperature ?) affects the action of remedies on the frog's heart the experiments were now repeated in January, that we might be able to compare the action of the chlorides with that of the iodides and bromides at the same time of the year.

Chloride of sodium.—As in the case of bromide of sodium, the effect on frequency was somewhat inconstant.

In one case, with the exception of trifling slowing, there was no effect on frequency, the beats remaining spontaneous to the end.

In two there occurred distinct inhibition quite of the potash type.

In a fourth marked slowing was produced, amounting in the end stages to inhibition, almost complete.

Lastly, in two cases the action of the ventricle became phasic, groups of beats alternating with intervals, this condition remaining to the end.

Here, though there was distinct disturbance of rhythm, spontaneous beats still persisted to the end. So that we have three cases of non-inhibition against three cases of inhibition.

Continuous faradisation, after arrest of the ventricle, gave the characteristic piling up.

The quantities required to arrest the heart were:

I.—Feb.	1.	Temp. of room	16°.	Quantity	16 c.c.
II.— „	1.	„	16·5°	„	14
III.— „	2.	„	—	„	20
IV.— „	4.	„	17°	„	23
V.— „	4.	„	17°	„	18
VI.— „	6.	„	9·5°	„	25
					116

Average 19·3 c.c. = 1·93 grms. = 29·77 grains.

Chloride of ammonium.—The effect on frequency was a slight increase of the contraction rate; the beats were accelerated on an average in the proportion of from three to five, three being the original frequency

In one case the beats became slightly slower throughout

but in this, as in all the others, spontaneous beats of good frequency persisted to the very last, *i.e.* till contractility disappeared.

When the heart had been arrested and single induction shocks caused no response, continuous faradisation was equally without effect.

This salt acts powerfully on contractility, the beats growing less and less till final cessation in diastole.

In all six experiments, as a first effect and for a short time the beats increased in height and became broader; in three this was very distinct, in the other three it was but slight; in the former there was also departure from the base line, showing a certain amount of persistent spasm.

The quantities required to arrest the heart were:

I.—Jan.	16.	Temp. of room	17° C.	Quantity	2·1 c.c.
II.— „	16.	„	18·5°	„	2·1
III.— „	17.	„	17°	„	2·
IV.— „	17.	„	16°	„	1·8
V.— „	18.	„	18°	„	1·8
VI.— „	18.	„	18°	„	1·8
					11·6

Average 1·9 c.c. = 0·19 grms. = 2·93 grains.

Chloride of potassium.—The first or second dose always caused slowing, then, in five out of the six cases, the beats again became more frequent, in some instances beyond the original frequency; then the beats again became slower with intervals, now amounting sometimes to from three to four minutes. This occurred on an average after 0·5 c.c. had been added. After as much as 0·76 c.c. = 1·17 grains, on an average, had been added, spontaneous beats ceased, though contractions could still be excited by induction shocks. This complete inhibition occurred when the beats were reduced respectively to $\frac{1}{3}$, $\frac{2}{5}$, $\frac{1}{5}$, $\frac{2}{5}$, $\frac{3}{4}$, $\frac{1}{6}$ of their original height.

When single shocks gave no result, continuous faradisation was equally without effect.

Contractility was completely destroyed after the addition of the following doses:

I.—Jan.	12.	Temp. of room	17° C.	Quantity	1·4 c.c.
II.— „	12.	„	17°	„	1·5
III.— „	13.	„	15·5°	„	1·8
IV.— „	13.	„	16·5°	„	1·7
V.— „	14.	„	19·5°	„	1·8
VI.— „	14.	„	18·5°	„	1·6
					9·8

Average 1·6 c.c. = 0·16 grms. = 2·46 grains.

In respect of the discussion of the results obtained, it may be mentioned that this discussion has assumed the cardiac muscular tissue to possess the property of rhythmic contractility apart from ganglionic structures.

Since the paper was written Dr. Gaskell's paper, read before the Royal Society, has reopened this question. The results here obtained, however, as also the discussion of these, are in nowise affected by this question as to rhythmic contractility necessitating two structures, nervous and muscular, or but one structure, viz. muscular. That which is alone required here is a rhythmically contractile tissue; this given, we here show that the action of drugs on such demonstrates the dissociation of that which underlies "*contraction rate*" or "rhythm," from that which underlies "*contraction height.*"

The actual results obtained have been so far alone given; of these, certainly the most striking is this—that given a rhythmically contracting tissue, the action of a drug on this may show itself in two directions:

1st. As affecting the intervals separating successive beats.

2nd. As affecting the actual value of the beats themselves.

Thus we have seen drugs affecting the frequency or contraction rate, and also the height of the individual beats (this last has been always referred to as the action on contractility). The stress has fallen now in one direc-

tion, now in another, but in every case there has been some effect in both directions.

It was mentioned at the outset that the ligature was not always exactly in the auriculo-ventricular groove; this, however, does not introduce an error, for the action of the drugs was tested on a rhythmically contracting tissue, and this in all cases was secured.

The question now arises, are the two manifestations above mentioned intimately connected one with the other, so that action on the one necessarily involves action on the other? or may they be more or less dissociated from one another, and separately subject to influence? The former supposition, viz. that of an intimate connection underlying the dual manifestation, requires for proof evidence of such union in the shape of constancy of relation, both in kind and degree, *i.e.* qualitative and quantitative. If closely united, the evidence of such must be that they move together in *some* definite direction at *some* definite rate.

On the other hand, the proposition that they are dissociated requires the negative evidence of *absence* of any constant relation, qualitative or quantitative—the demonstration that they do not move together.

The consideration of this subject necessitates the use of *terms* in place of the phrases "value of each beat," "frequency of contraction." For the former the word *contractility*, as naming the underlying element, may be substituted; for the latter the term "*excitability*" has been selected. The term is not without objection, and must be taken in the wide significance of "*conditions antecedent to the contraction.*" These conditions are, of course, causal, but whether they be of the nature of "conditions generating the stimulus," or "conditions preparing the contractile tissue for the stimulus, *i.e.* rendering the stimulus available," must be left undiscussed. The terms being thus defined, we have to consider whether *excitability* and *contractility* must be associated or may be dissociated.

What do we actually find? In one case we note

increased frequency attending increased height of beat, in another increased frequency attending diminishing height; and examples of this are to be found not only in the case of different hearts, but even in the same heart it may be found. Thus the ammonia salts, *e.g.* the chloride or bromide, frequently show a primary increase of frequency even during the stage of increased height of beat, and this increased frequency is not only maintained but may even go on increasing as the height of the trace steadily falls. Thus, there is no constant qualitative relation between *contraction rate* and *contraction height.*

Again, in one and the same heart we note as the effect of a drug at one time marked effect, say on excitability, with no appreciable effect on contractility; a little later, and with no noticeable effect on excitability, the stress now falls on contractility.

Potash salts illustrate this very well. Thus, the first additions as a rule are followed by slowing, to the extent may be of doubling the length of the intervals, with scarcely any effect on the height of the beats; a little later on, and with almost unchanged rhythm, the beats may diminish by two thirds within the short space of seven beats.

Thus, then, even the same heart shows no quantitative relation that is constant between excitability and contractility.

One might multiply evidence in the same direction. Thus, quite suddenly a heart, beating at intervals of fifteen to twenty seconds, will start off at a rate five to ten times as fast, with little if any variation in height of beat. But sufficient has been said to prove that, whatever underlies these two manifestations, which are measured by *contraction rate* and *contraction height*, they may be separately influenced, and therefore so far are distinct.

Hence one may speak of a drug as acting on either *excitability* or *contractility*, and the results already given may be more briefly and clearly summed up as follows:

In the case of the drugs already examined, and probably in all cases, the action is not exclusively on either *excitability* or on *contractility; both* are affected.

The degree, however, in which one or other suffers varies with different drugs. Thus, with regard to the salts of the three different bases, sodium, ammonium, potassium, the effect on excitability varies greatly; and, whilst potassium salts strongly affect excitability, sodium and ammonium salts affect excitability relatively but slightly. And thus, whilst with potassium salts it was the exception not to get permanent arrest of spontaneous beats before contractility was destroyed, with sodium and ammonium salts it was the exception when spontaneous beats did not continue up to the very end, and, moreover, with a final frequency little short, often in excess, of the original frequency.

Though there is this broad division into potassium salts on the one hand and ammonium and sodium salts on the other, the salts of ammonium and potassium form the extremes, those of sodium being intermediate, affecting as they do excitability rather more than ammonium. As to the action on contractility, the quantities of the drugs used constitute the measure of this action, since in each case the contractility was reduced to *nil*. On examining the table of quantities it will be seen that a very different relation now obtains. Potassium and ammonium come very close together, whilst sodium is widely separated. The two former are so near numerically that it would be unsafe to draw inferences from the differences in the actual numbers; in passing to sodium the highest estimate, in the case of the chloride, bromide, and iodide, would give the relation as one to ten, the sodium salts being one tenth as poisonous as those of ammonium and potassium.

Table of quantities.

	Quantity in grs.
Potassium hydrate . . .	1·35

	Chloride. Quantity in grs.	Bromide. Quantity in grs.	Iodide. Quantity in grs.	Relation of molecular weights.
Sodium .	29·77	51·7	84·24	58·5 : 103 : 150
Ammonium	2·93	3·7	6·3	53·5 : 98 : 145
Potassium	2·46	3·28	3·16	74·5 : 119 : 166

Citrate.		Molecular weights having equal number of atoms of base.
Sodium . .	16·66 ...	357
Ammonium .	3·3 ...	339
Potassium .	2·46 ...	324

In the above the only column which perhaps requires explanation is that giving the relation of the molecular weights. The object of this was to see, if, taking a given base, any relation could be traced between the action of its salts as here tested and the molecular weights of the same salts.

There does appear to be some such relation in the case of the sodium salts of chlorine, bromine, and iodine. But this fails in the case of the ammonium and potassium salts, and so, the results obtained do not point in any one direction. Obviously, however, a very much larger number of experiments would be needed to arrive at any definite conclusions, positive or negative, on this point.

One must be careful not to take effect on contractilty as the exclusive measure of poisonous action.

Arrest of the contractions rather should be taken to represent the poisonous action, and this we have seen may happen in two ways, by the action on *contractility*, and by the action on *excitability*. Hence though as to the first ammonium and potassium show but little difference, as to the latter they diverge widely, and potassium stands as by far the more powerful poison.

The recognition of the above has especial importance from a clinical standpoint, for, if we be dealing with the functions of an organ, the arrest of such functions concern us more immediately than the precise mode in which such arrest is effected. And if a drug threaten in two directions, the dosage must take both of these into account.

Accordingly, represented in descending order, we have :

Potassium salts, most poisonous, both *excitability* and *contractility* powerfully affected.

Ammonium salts, next in order, *contractility* suffers almost alone.

Sodium salts, least poisonous, *contractility* suffers chiefly, but *excitability* is more affected than in the case of ammonium salts.

Further, it must be remembered that sodium salts are not only least poisonous of the three, but are, indeed, very weak poisons as compared with both potassium and ammonium salts.

The iodides and bromides of potassium and ammonium are so largely used that the importance of the foregoing is apparent if no further conclusion than this be drawn :— *that, the action on one tissue being selected and all other conditions being kept as far as possible identical, if one drug prove itself more active than another, it is at least not improbable that this same drug will also prove itself more active under the more complex conditions presented by the organism as a whole.*

A very guarded conclusion is obviously the only one that can be drawn when one passes as here from simple to very complex conditions, but the above conclusion is surely warranted.

Hence these experiments would suggest the substitution of the bromides and iodides of sodium for those of ammonium and potassium, and the use of those of ammonium preferably to those of potassium ; but the very wide gap separating sodium salts from both ammonium and potassium points especially to the use of the first, and the more so that, so far as clinical evidence goes, it is to the effect that, therapeutically, the salts of sodium and ammonium are as powerful as those of potassium.

In conclusion, one or two points may be touched upon. The very slight degree of poisonous action of the chloride, bromide, and iodide of sodium, was such that very large quantities of the drugs had to be added, so much so, that physical changes, such as osmosis, became probably important factors in the arrest of the heart. This, of course, would not affect the previous statements, but would rather

place still lower in the scale of poisonous action the above sodium salts, for clinically, the doses given would never even approach those here used, so that physical conditions would scarcely become factors in their therapeutic action.

These physical conditions may, however, account for the peculiar occurrence of piling up under the influence of continuous faradisation, which phenomenon was constant for the above sodium salts, and which Fig. VII illustrates.

It will be seen further on that with sodic citrate, of which salt considerably smaller quantities were used, this phenomenon did not appear.

Another point which touches a subject of very great importance may also be noticed. Looking over the results obtained, it will be seen that the chlorides, bromides, and iodides of potassium closely resemble each other in their action ; the same is true of the ammonium salts. The sodium salts are best not considered here, since the physical elements introduced might obscure any such relation existing. The facts then to be put together are :—that salts resembling each other in having a common base resemble each other in their action.

The statement, that of a series of salts with the same base, the poisonous action is dependent on the base is not new, certainly with regard to potassium salts. These experiments, so far as they go, tend to confirm this statement, which, however, can scarcely represent the whole truth, for looking back at the table of quantities, it will be seen that the citrate of potash is as poisonous as the chloride, and rather more so than the iodide and bromide ; it will be said, this is in direct opposition to clinical evidence.

It would be so, if one is to hold that the frog's heart as here used takes into account the whole action of the drug, but, if a salt do not act as a whole but individual elements composing it keep their identity, so far as action goes, then one must assume that one side or aspect of the drugs examined is here left out, and that the test is an imperfect one.

This again does not invalidate the conclusions drawn, for, given the bromide, say of three different bases, and that the basic element alone is taken account of, one must assume that the unaccounted-for bromine element being constant in all three, may, after the manner of all constants, be disregarded, and the salts be represented *relatively* in terms of their differences, *i.e.* of their bases.

The Citrate Group.

These organic salts were chosen in order to contrast them with the very definite group of the iodides, bromides, and chlorides amongst inorganic salts.

Citrates of soda, ammonia, and potash.

Of soda and potash the tribasic salts were employed, of ammonia the di-ammonic salt.

With reference to the quantities used, an important element is the water of crystallisation, which varies in the above three salts. Thus:

The molecule of tri-potassic citrate contains one molecule of water of crystallisation	$K_3C^6H_5O_7 + 1Aq.$	Mol. wt. =	324
That of tri-sodic citrate, 5·5 of water, the crystals having the composition .	$2Na_3C^6H_5O_7 + 11Aq.$	„	$= (357)_2$
That of di-ammonic citrate, none .	$(NH_4)_2HC^6H_5O_7$	„	= 226

The quantity of ammonic citrate which would contain the same number of atoms of the radical ammonium as the sodium and potassium salts, *i.e.* three, would be represented by the number 339. So that approximately equal weights of all three salts would contain the same number of atoms of potassium, sodium, ammonium.

Tri-sodic citrate.—This salt affects frequency but slightly, and so long as the contractions are visible, rhythm is manifest.

One case out of the six was somewhat exceptional, here

the effect on frequency was much more marked in the way of slowing, though even here in the final stages there reappeared a faint waviness of the trace, indicating faint spontaneous contractions.

The effect of continuous faradisation was certainly not diminished, if anything, it was increased. Even after the beats had completely ceased or had been reduced to a minimum, complete tetanus, though of a low altitude, was obtained.

In no case was the piling up obtained, which the chlorides, bromides, and iodides of sodium showed, when continuous faradisation was applied after the heart had been arrested by the drug.

The stress, as a rule, fell early on contractility, the contractions growing feeble rather rapidly, whilst rhythm suffered but slightly. In four out of the six cases, and doubtfully in a fifth, a certain amount of recovery took place under the action of the drug, *i.e.* the beats after reduction to a certain point, increased again slightly in spite of continued addition of the drug. The recovery was not, however, of long duration.

The early and sudden action on the height of the trace is noteworthy, in five out of the six cases it was marked, so that by far the larger amount of the drug was spent in destroying the small residuum of contractility remaining after the above primary effect.

The quantities employed were:

I.—Dec.	31.	Temp. of room	8°.	Quantity	16 c.c.
II.— „	31.	„	8·5°	„	14
III.—Jan.	2.	„	11°	„	12·5
IV.— „	2.	„	12·5°	„	13
V.— „	6.	„	17°	„	5
VI.— „	6.	„	17°	„	5
					65·5

Average 10·83 c.c. = 1·083 grms. = 16·66 grains.

Citrate of ammonia $(NH_4)_2HC^6H_5O_7$.—In all six experiments spontaneous beats occurred up to the very end,

i.e. so long as contractility remained. As to quickening or slowing no constant effect was produced, but in two, out of the six, an effect similar to that caused by potash obtained, viz., a rather sudden slowing of the rhythm, with no appreciable change in contractility. As was the case with soda, a certain amount of recovery occurred after the beat had been primarily reduced.

With respect to faradic excitability, here, as with the soda salt, there was certainly no diminution, at any rate so long as the height of the beats were of sufficient value to give definite results in this direction. If anything, the change was towards increase.

When the contractility had been destroyed by the drug and single stimuli were without influence, continuous faradisation was equally without effect, *i.e.* no piling up obtained.

In four out of the six cases there was distinct primary increase in height of beat, in one it was doubtful, in one the only effect was a broadening of the beat. The stage of increase was of but short duration. The recovery before mentioned occurred in four out of the six cases; it took place after reduction of the beat to a very small quantity, and was but very slight. The decline in the height of the beats was gradual in some, in others occurred rather rapidly.

Quantities:

I.—Dec.	30.	Temp. of room	10°.	Quantity	2·4 cc.
II.— „	30.	„	10°	„	3·3
III.—Jan.	4.	„	15°	„	3
IV.— „	4.	„	16°	„	4
V.— „	5.	„	17°	„	3·9
VI.— „	5.	„	18°	„	3·4
					20·0

Average 3·3 c.c. = 0·33 grms. = 5·09 grains.

Citrate of potash.—The effect of this salt was primarily to slow the rhythm. This set in early, appearing as a rule after the first dose, though it often went on increasing with the first few doses. As a rule, this early effect was

without a corresponding effect on the height of the beat; thus the rate might be reduced by one half with little noticeable effect on the height of the trace.

Complete inhibition did occur in five out of the six cases, but not till the beat had been greatly reduced, viz. to $\frac{1}{4}$th, $\frac{1}{5}$th ($\frac{1}{5}$th—$\frac{1}{6}$th), $\frac{1}{5}$th, $\frac{1}{5}$th respectively, of the original height of the beat. This effect, then, was much less prominent than was the case with the chlorides, bromides, and iodides of potassium. The primary slowing above noted did not go on increasing at the same rate, and as a rule a stage followed in which great reduction in height occurred with scarcely appreciable change of rhythm.

Faradic excitability was markedly diminished, and a current giving strong tetanus before addition of the drug, finally failed to give any, or indeed only an initial contraction.

When the heart had been arrested by the drug and single shocks caused no effect, continuous faradisation was equally without effect.

There was no primary increase in the height of the beat, but decline from the very commencement; but whilst in the early stage the stress appeared to fall on rhythm—the decline in height being very slight—later on the effect on contractility became marked, the trace in some falling rapidly without corresponding effect on rhythm.

Quantities:

I.—Dec.	29.	Temp. of room	7°.	Quantity	0·9 c.c.
II.— „	29.	„	7·5°	„	1·8
III.—Jan.	3.	„	15°	„	1·8
IV.— „	3.	„	16°	„	0·9
V.— „	7.	„	14°	„	2·5
VI.— „	7.	„	14°	„	1·8
					9·7

Average 1·6 c.c. = 0·16 grm. = 2·46 grains.

The results obtained from the citrate group may be thus summarised:

Sodium and ammonium salts affect *excitability* but slightly; they arrest the heart by destroying *contractility*.

Both appear, if anything, to increase *faradic excitability*.

Specially it must be noted, that when the heart has been arrested by sodium citrate, continuous faradisation is without effect, *i.e.* no piling up obtains. And, further, that the sodium citrate is much more poisonous than the sodium salts of the chloride group.

With respect to ammonium citrate, note that a primary increase in the height and breadth of the trace is the rule.

Potassium citrate affects both *excitability* and *contractility*, but the effect on excitability is much less marked than is the case with the potassium salts of the chloride group; still, the effect was decided, especially in the end stages.

There is nothing special as to the action on contractility. Faradic excitability is diminished from the commencement.

Comparing these results with those of the chloride group, we note the agreement of the sodium and ammonium salts, in the slight effect on excitability, and in the mode in which they arrest the heart.

They contrast, however, in their effect on faradic excitability; thus, whilst the chloride group lessens, the citrates appear to increase this.

In respect of sodium citrate, the two points specially mentioned, viz. relatively small dose and absence of piling up go together, and are of importance in relation to the chloride group, since here we had large dosage, and in all cases the final mounting up of the trace under continuous faradisation. The citrate results are in conformity with the view that this mounting or piling up indicates physical change in the muscular tissue.

In addition to the agreement above noted in respect of the effect on excitability and contractility, the ammonium citrate agrees with the ammonium salts of the chloride group in its primary effect of increase in height and breadth of trace. The only point of contrast hence is in respect of faradic excitability.

The potassium citrate contrasts with the potassium salts of the chloride group in its much slighter action on *excita-*

bility. It is true complete inhibition did obtain in five out of the six cases, but the beat was greatly reduced before such occurred.

In its influence on faradic excitability, there is agreement with the chloride group.

The doses required to destroy contractility are for both the ammonium and potassium citrates about the same as required in the chloride group. This question of poisonous action has been discussed with reference to clinical experience.

In the case of the salts of potassium we have the most complete series. Thus we have examined the hydrate, the chloride, bromide, iodide, and the citrate.

Taking the hydrate as the starting point, we note here—

1st. The tendency to produce *persistent spasm.*

2nd. The tendency to produce *inhibition.*

3rd. The tendency to lessen *faradic excitability.*

Passing to the chloride group, we note that the tendency to produce persistent spasm has disappeared, that the action on spontaneous excitability is, however, still very marked, also that on faradic excitability.

Passing to the citrate of potassium, we note the absence of any tendency to produce persistent spasm, that the action on spontaneous excitability, *i.e.* the tendency to inhibit, is much diminished ; that the action on faradic excitability still remains, though it is difficult to state comparatively the degree in which it persists.

With the exception, then, of the persistent spasm feature, we note a similarity in the nature of the action running through all these salts of potassium.

The ammonium series is less complete since the influence of the hydrate on the spontaneous working of the heart could not be so completely tested.

Here we note, however, that the tendency to produce persistent spasm runs through all the salts of ammonium ; though in the chloride group and in the case of the citrate, this tendency is but slight, appearing early and for only a short period.

Further, amongst the salts of ammonium there is agreement in the negative quality of slight action on rhythm.

As to the effect on faradic excitability there is no agreement, for whilst ammonia increased the above the iodide lessened it, whilst the citrate increased it. (The chloride and bromide were not examined.)

The sodium salts can be less easily compared, for in the chloride group, physical action probably comes into play. The persistent spasm which the hydrate excites does not appear with any of the salts. The action on spontaneous excitability is slight as compared with potash salts, and in this respect there is agreement throughout the sodium salts. The action is, however, more marked than in the case of ammonium salts. As to the effect on faradic excitability, the sodium salts show as little constancy of action as those of ammonium; thus, whilst soda increases faradic excitability the iodide lessens it, whilst the citrate shows, if anything, a tendency towards increase.

EXPLANATION OF DIAGRAMS.

Diagram 1.

Fig. I.—Illustrating "genesis of tetanus" by fusion of beats, the result of continuous faradisation.

A, B, C, show the effect of increasing the strength of the current, the frequency of interruption of the primary current remaining the same.

The figures above the lines represent the positions of the secondary coil.

D, represents a later stage.

As the strength increases from 7·5—7—6, it will be seen that the more complete tetanus also corresponds with the greater number of contractions. In D the fusion is so complete that individual beats are no longer countable.

Fig. II.—Shows effect of sodium hydrate on the excitability of the ventricle to continuous faradisation.

Nov. 25. Temperature of room 16° C.

A. Before the addition of sodic hydrate.

B. After 25 minims of a 1 per-cent. solution.

C. After 75 „ „ „

Fig. III.—Shows similarly the effect of ammonium hydrate.

Nov. 26. Temperature of room 15·5° C.

A. Before the addition of ammonium hydrate.

B. After the addition of 10 minims of 1 per-cent. solution.

} see Diagram 2.

C. „ „ 24 „

D. „ „ 55 „

Fifty-six minutes from first dose.

Diagram 2.

Fig. III, A. and B. (see above).

Fig. IV.—Shows effect of potassium hydrate. A 10 per-cent. solution was here used.

Feb. 12. Temperature of room 16° C.

A. Before addition.

B. 10 minutes later after 0·15 c.c. = 2·55 minims. The tetanus is here taken in the earliest stage of the potash effect, note the faint evidence of diastolic rise.

C. 10 minutes from B, after 6 minims in all. Note the diminution of effect, together with the presence of considerable amount of persistent spasm.

D. and E. are after 7·6 minims and 9·3 minims respectively, they show progressive decrease in excitability.

Fig. V.—Jan. 28. Temperature, 16·5° C. Bromide of ammonium, 10 per-cent. Chosen as typical of the action of ammonium salts of this group, viz. chlorides, bromides, iodides.

A. Shows effect of first addition of 0·2 c.c. = 3·4 minims also a few beats before the addition.

B. About five minutes from first addition; 0·6 c.c. = 10 minims have been added.

C. 10 minutes from B, 1·8 c.c. = 30·5 minims have been added.

D. 2 minutes from C, 2·2 c.c. = 37·3 minims „

DIAGRAM 3.

E. Comes immediately after D, 2·4 c.c. = 40·6 minims was the total quantity added.

Note certain amount of persistent spasm in A and B, the beats are too frequent for diastolic contraction to appear between the individual contractions.

Note slight irregularity preceding the end stage.

FIG. VI.—Jan. 19. Temperature 15° C. Bromide of potassium, 10 per cent.

A. First dose 0·1 c.c. = 1·7 minims.

B. 4—5 minutes later, after 0·3 c.c. = 5·07 minims

C. 2—3 minutes later, fifth dose = 0·5 c.c. = 8·45 minims

D. 3 minutes later. The last spontaneous beat is here represented, viz. after 0·8 c.c. = 13·52 minims, so that hence on there was complete inhibition.

E. 10 minutes later, 1·1 c.c. and 1·2 c.c. (18·6 and 20·3 minims) = the quantities added. The beats are the result of excitation.

F. 8—9 minutes later, after 1·4 c.c. = 23·7 minims.

G. 5 minutes later, final stage, 1·7 c.c. = 28·8 minims, the total quantity added.

Note the primary slowing in B and C, the rhythm was further somewhat irregular; in D, just before complete inhibition, there was considerable irregularity. The subsequent charts show that, though inhibited, contractility still remained, the figures above the beats represent the positions of the secondary coil.

FIG. VII.—Showing effect of continuous faradisation after heart has been arrested by bromide of sodium. The charts are typical of the chlorides, bromides, and iodides alone.

A. Jan. 24. Temperature 17·5° C. Sodium bromide 10 per-cent. The effect of continuous faradisation with the secondary coil at 4, and at 0 (pushed home) is shown. The previous want of effect of single shocks is shown, and of continuous faradisation at 6.

B. Jan. 27. Temperature 17° C. Sodium bromide 10 per-cent.

Similar to above, this perhaps represents the more usual degree of effect produced.

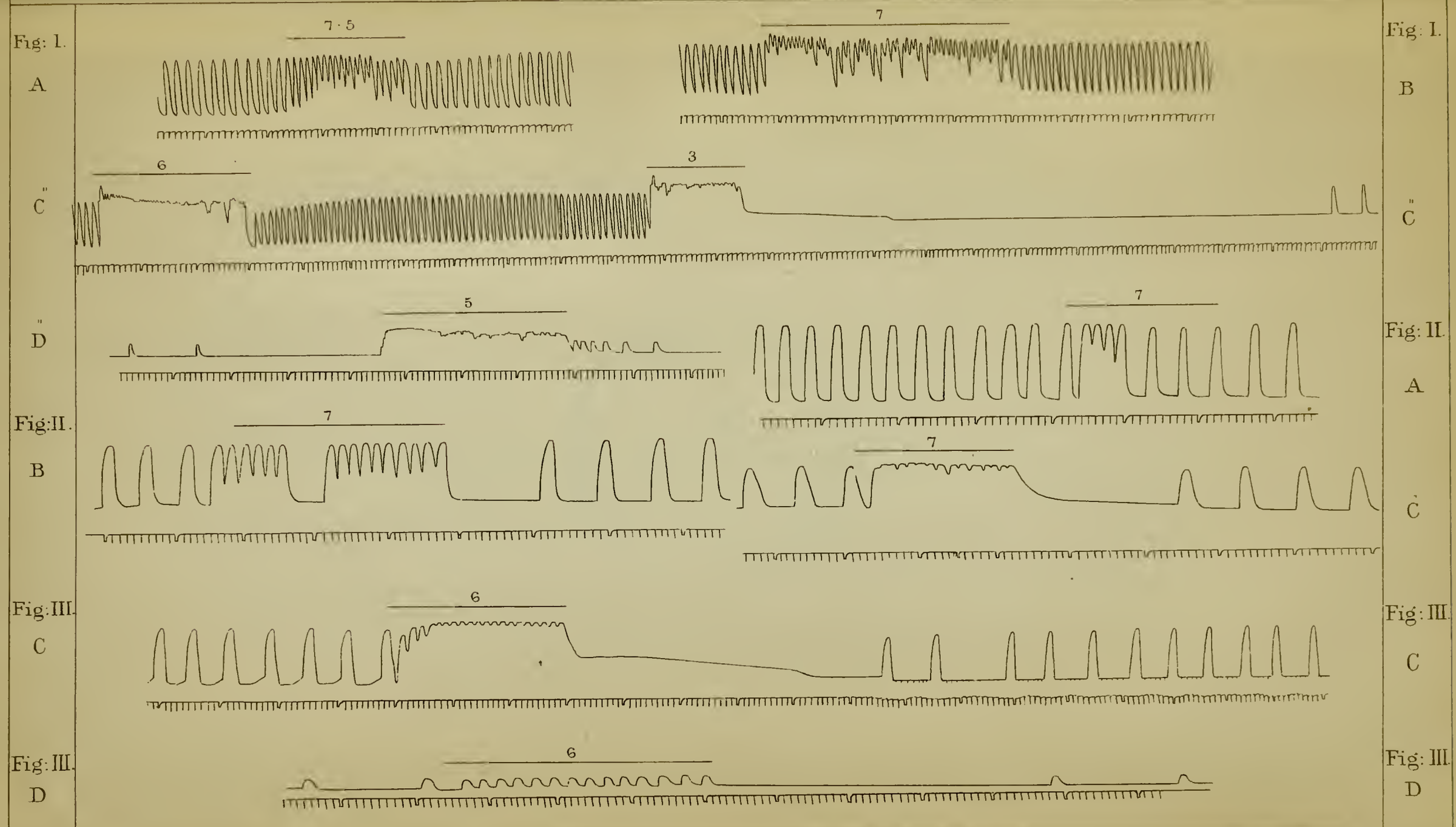
Fig: I.
A
7·5
7
B
6
3
C"
C"
5
7
D"
Fig: II.
A
Fig:II.
7
B
7
C
Fig:III.
6
C
Fig: III.
C
Fig:III.
6
D
Fig: III.
D

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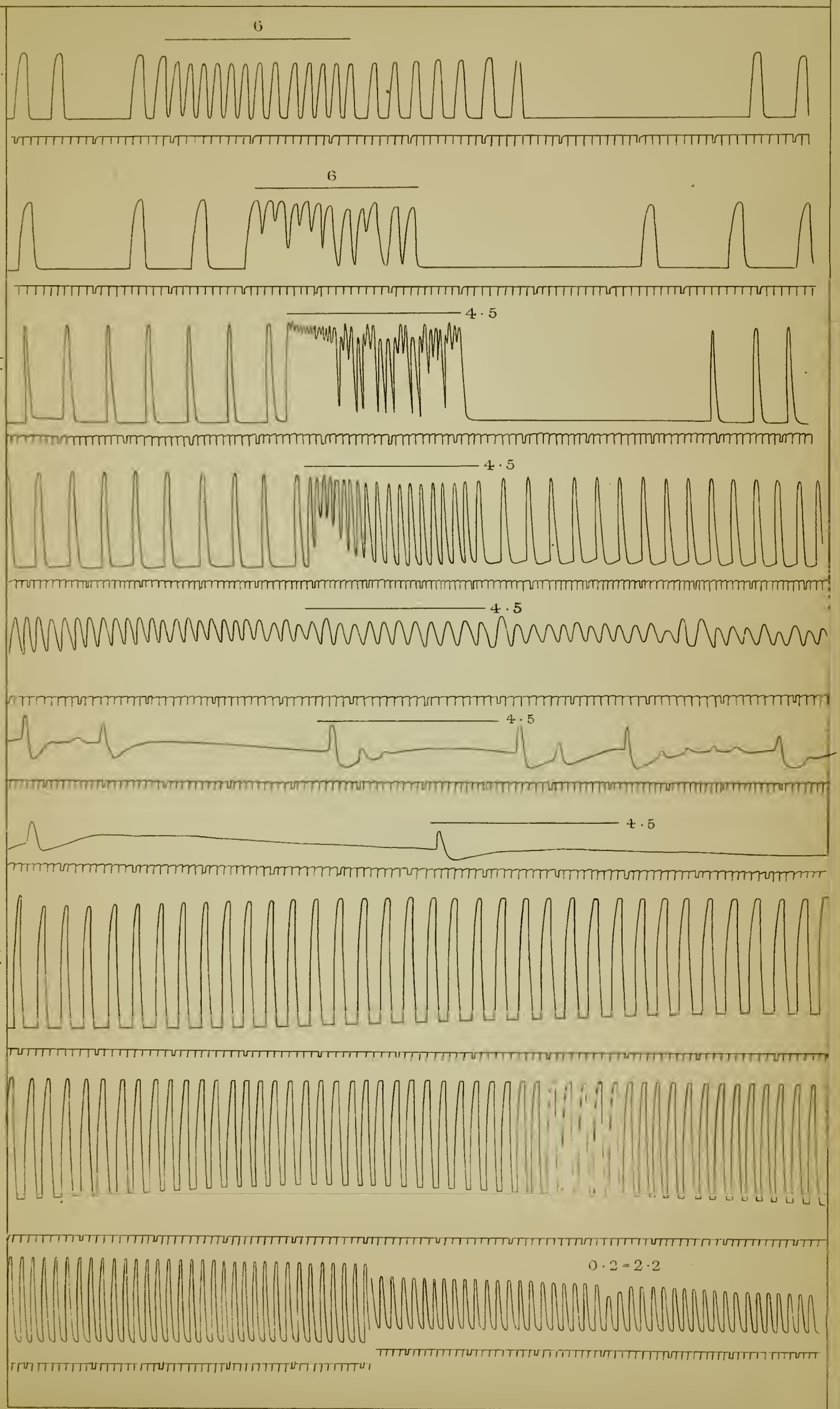

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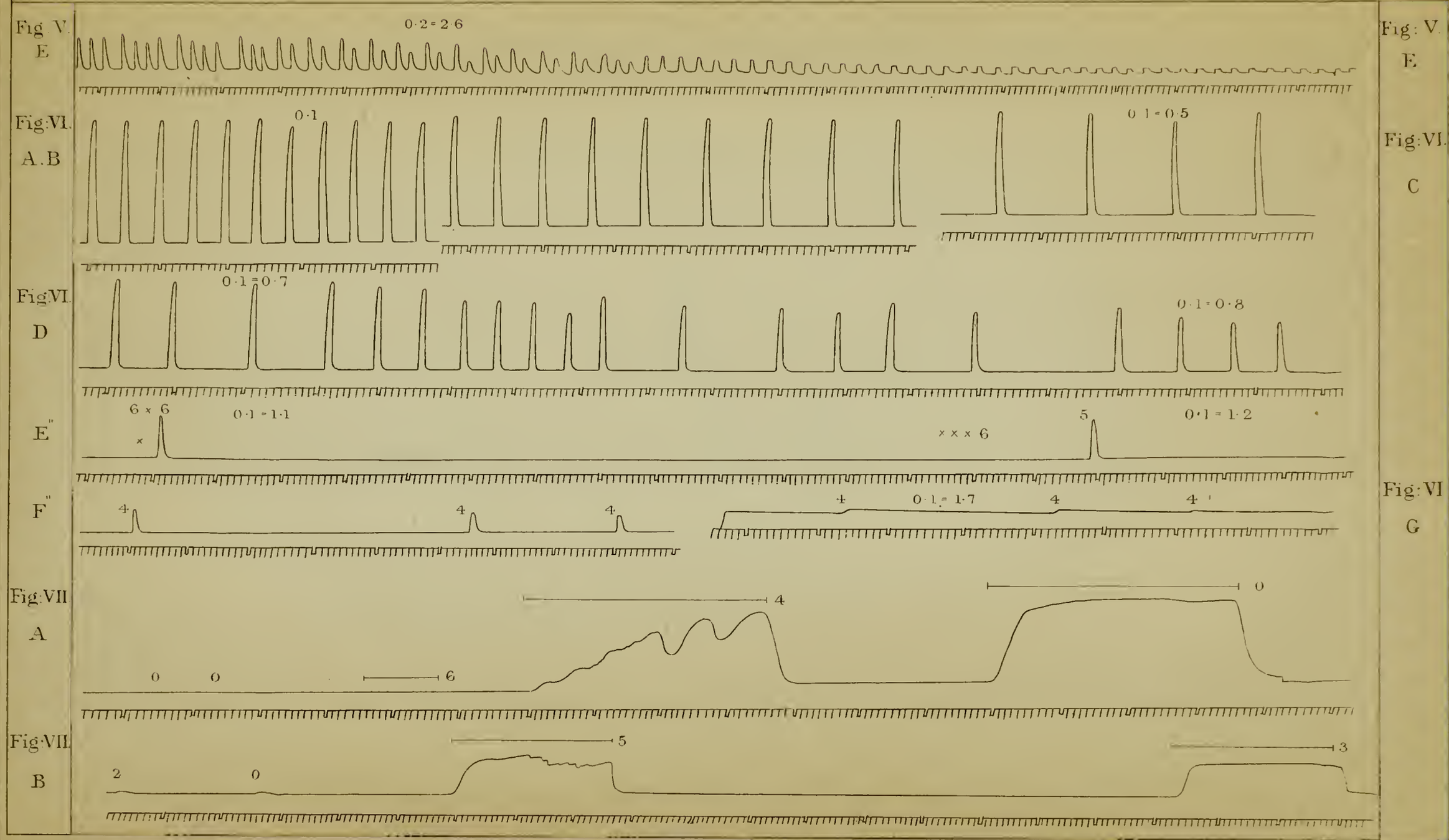

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